***Amendments to the Specification***

Please amend the title at page 5, line 13 as follows:

**Description of the ~~Figure~~ Figures**

Please add below the title "Description of the Figure" at page 5, line 13, the following new paragraph:

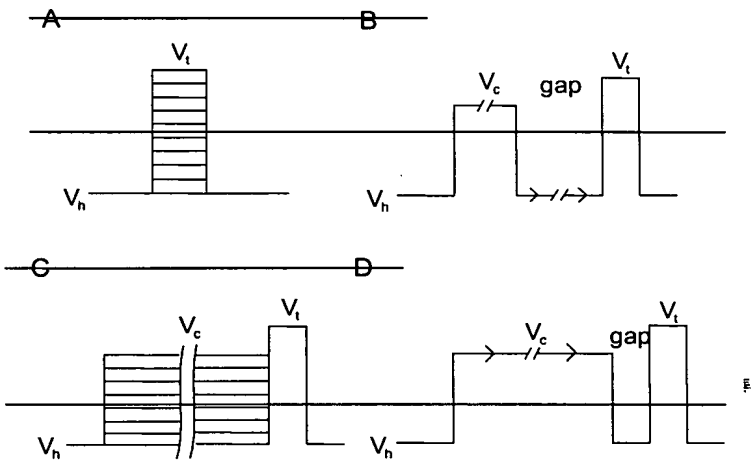
FIGS. 1A, 1B, 1C, and 1D are voltage pulse protocols used to assess the potency and kinetics of inhibition of the $Na^+$ channels by the compounds as follows: FIG. 1A: IV-curves, FIG. 1C: steady-state inactivation, FIG. 1B: repriming kinetics, and FIG. 1D: time course of binding.

Please replace the paragraph starting at page 5, line 15, with the following paragraph:

FIG. [[1]] 2 depicts a graph showing the synergistic antiallodynic effect of gabapentin and the sodium channel blocker 4-(4'-fluorophenoxy)benzaldehyde semicarbazone (Co 102862) in the Chung model of neuropathic pain in rats (Kim and Chung, *Pain 50*: 355-363 (1992)).

Please replace the paragraph at page 28, line 3 through page 29, line 8, with the following paragraph:

The following voltage pulse protocols **A, B, C, and D** are used to assess the potency and kinetics of inhibition of the $Na^+$ channels by the compounds (~~Fig. 1~~ FIGS. 1A-1D).




~~**Figure 1.** Voltage pulse protocols. **A.** IV curves. **C.** Steady-state inactivation. **B.** Repriming kinetics. **D.** Time course of binding.~~

Current-voltage relationship (IV-curve), protocol **A** (FIG. 1A), is used to report the voltage at which the maximal inward $Na^+$ current is achieved. This voltage is used throughout the experiment as testing voltage, $V_t$. The steady-state inactivation (or, availability) curve, protocol **C** (FIG. 1C), is used to get the voltage at which almost complete (≥95%) inactivation of $Na^+$ channels occurs; it serves as voltage for conditioning prepulse, $V_c$, throughout the experiment. Protocol **B** (FIG. 1B) reports how fast the channels recover from inactivation at hyperpolarized voltages. This permitted us to set up the duration of the hyperpolarization gap which is used in measurement of the kinetics of binding of compounds to inactivated $Na^+$ channels (protocol **D** (FIG. 1D)). Channel repriming under control conditions is fast (≥90% recovery during first 5-10 ms). If a drug substantially retards the repriming process, then it becomes possible (protocol **D**) to accurately measure the kinetics of binding of the inhibitor to inactivated channels as well as the steady-state affinity ($k_+$ and $K_i$). To estimate $k_+$ values, the reduction in peak currents in successive trials with varying pre-pulse duration is plotted as a function of pre-pulse duration and the time constant ($\tau$) measured by mono-exponential fit. A plot of $1/\tau$ as a function of antagonist concentration then allows calculating of the macroscopic binding rates of the antagonists. To determine $K_i$ values the partial inhibition curves measured by fractional responses in steady-state are fitted with the logistic equation:

$$I/I_{control} = 1/(1 + ([antagonist]/K_i)^p), \qquad \text{Eq. 2}$$

where $I_{control}$ is the maximal $Na^+$ current in the absence of antagonist, [antagonist] is the drug concentration, $K_i$ is the concentration of antagonist that produces half maximal inhibition, and p is the slope factor.

Please replace the paragraph at page 32, line 27 through page 33, line 2, with the following paragraph:

The tactile antiallodynia effect of Co 102862 and gabapentin was tested alone or in combination in the Chung model of neuropathic rats. As shown in FIG. [[1]] 2, rats that received 1.25 mg/kg Co 102862 p.o. showed moderate antiallodynia effect whereas 25 mg/kg gabapentin s.c. exhibited minimum or no effect when given alone. However, when both compounds were given together, a much greater withdrawal threshold was observed than if one were to add effect of Co 102862 and gabapentin given individually. Thus, the combination of the two drugs has a synergistic effect. See FIG. [[1]] 2.